chloride uptake occurs in the affected areas.[31] Consequently, its use in this case was ideal and the results obtained were gratifying.

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Infective Endocarditis Caused by Moraxella kingae

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THE ROLE OF Moraxella kingae and closely related Gram-negative, pleomorphic bacilli (of the genera Neisseria, Bacteroides, Brucella, Hemophilus, Actinobacillus and Pasteurella) in human disease needs further definition. One previous case of endocarditis caused by Moraxella kingae or new species I has been reported in a 4-year-old child with a ventricular septal defect.[1] The following case documents infective endocarditis due to this organism in an adult.

Report of a Case

A 28-year-old man, a previously healthy lifeguard, had sudden onset of shaking chills, temperatures to 40°C (104°F), myalgias and headaches on June 23, 1976, three days after an abrasion on the right great toe. On June 27, a severe pain in the right femoral region lasted for several hours. On July 1, a half-hour episode of expressive aphasia during temperature elevation to 40°C led to hospital admission. A cardiac murmur was not noted on two previous physical examinations. The patient jogged and swam regularly and in competition without any apparent difficulty. He did not use drugs.

Abnormal findings on physical examination were: a flushed, acutely ill appearance; scleral injection and a hairline abrasion on the dorsum of the right great toe. Blood pressure was 130/80 mm of mercury, pulse 100 and regular, temperature 37°C (98.6°F). A right subconjunctival

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hemorrhage and splinter hemorrhage on the right index finger appeared three days later. On the seventh hospital day, multiple petechial hemorrhages appeared on the patient's trunk and a grade I/IV blowing diastolic murmur was heard for the first time at the cardiac apex, left sternal border and right second intercostal space. There was intermittent tenderness in the left costovertebral angle, but no splenomegaly was noted. During this first week in the hospital, the patient had daily shaking chills with temperature elevations to 40.5°C (104.9°F).

On July 1, hemoglobin was 13.1 grams per dl, leukocyte count was 11,500 with 70 percent neutrophils, 4 percent bands and 15 percent monocytes. Sedimentation rate was 31 mm per hour. Urine sediment contained three leukocytes per high power field. Cultures of six blood specimens, drawn during the first 48 hours in the hospital, grew no organisms. Culture of a blood specimen drawn on the fifth hospital day was reported on the seventh hospital day as growing a pleomorphic Gram-negative organism. Subsequently cultures of seven blood specimens drawn after the fifth hospital day grew the same organism which was ultimately identified as Moraxella kingae. This identification was confirmed by the bacteriology laboratories of the Los Angeles County and State of California Departments of Health and the Center for Disease Control. Cultures of cerebrospinal fluid, bone marrow, urine, liver tissue and stool were negative. Several chest films, a gallium scan and an intravenous pyelogram showed no foci of infection. The latex agglutination test for rheumatoid factor was negative in the second and fourth weeks of illness. Six impacted molar teeth, including two extra third molars protruding into the maxillary sinuses, were extracted while the patient was receiving antibiotic therapy during the fifth week in hospital. No abscess was noted. Gingival tissues showed evidence of chronic inflammation.

The isolated organism proved to be very sensitive to penicillin (minimal inhibitory concentration 0.12 μg per ml, minimal bactericidal end point 0.24 μg per ml). The organism was also sensitive to ampicillin, chloramphenicol, cephalothin, erythromycin, kanamycin, tetracycline and methicillin. Excellent bactericidal levels were obtained with intravenous administration of penicillin G, 5 million units every six hours (1:128 four hours after the penicillin infusion). Because of the slow growth characteristic of this organism, however, the end point with this technique was not ideal.

The patient was maintained on 20 million units of penicillin daily for 28 days. He was afebrile after 72 hours of treatment with antibiotics. The intensity of the diastolic murmur did not change during treatment and congestive heart failure did not develop. Blood cultures were negative two and four weeks after therapy with penicillin was discontinued.

The patient remains well on follow-up examinations. No abnormality of the aortic or mitral valve leaflets is evident by echocardiography, although the patient clearly has the murmur of aortic regurgitation on auscultation and a slightly widened pulse pressure of 120/60.

Comment

The Center for Disease Control has 40 isolations from human specimens in its files on Moraxella kingae: 15 blood cultures; 9 throat cultures; 6 bone or joint aspirates, and 10 specimens from miscellaneous sites (personal communication, Dr. R. Weaver). The clinical disease associated with the samples is not on record for most specimens.

In man, the Moraxellae reside in mucous membranes of the eye, nasopharynx and genitourinary tract.[2] Moraxellae have been identified as causative agents of angular conjunctivitis since the late 19th century and more recently have been associated with septicemia,[3] purulent pericarditis,[4] septic arthritis,[5] meningitis,[6] endocarditis,[1,7] endophthalmitis[8] and corneal abscess.[9]

Moraxella kingae organisms are pleomorphic Gram-negative rods and sometimes produce pitting on blood agar. There is some question as to whether Moraxella kingae should really be classified as a member of the Moraxella group, primarily because M. kingae is saccharolytic, while other members of this group are nonsaccharolytic.[10]

It is possible that this patient's infection originated in the inflamed mucosal surfaces adjacent to the impacted molar teeth or in the toe abrasion that occurred three days before the onset of fever. The explanation for why a fastidious organism of low-grade pathogenicity might cause endocarditis on a normal valve is not apparent at this time. In the presence of known antecedent cardiac valvular disease or a heart murmur at the onset of illness, the pathogenic organism would not have been identified. Antibiotic therapy would have

been started, as is customary, after the initial blood cultures, without necessarily waiting for a positive culture report. We can only speculate that fastidious organisms, similar to Moraxella kingae, are pathogens in cases of culture negative endocarditis.

Summary

In a 28-year-old man without antecedent heart disease or a drug habit, infective endocarditis caused by Moraxella kingae led to aortic regurgitation. Intravenous administration of penicillin G (20 million units per day) for 28 days cured the infection. Six initial blood cultures were negative, delaying recognition of the pathogen.

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Aspirin Ingestion and Gastrointestinal Bleeding in Children

THE PROBLEM of gastrointestinal bleeding has changed somewhat in the past few years. If one looks at the standard textbooks on gastrointestinal bleeding in children, practically all of them indicate that in approximately 40 percent of the instances the cause is idiopathic or unknown. Those figures have changed nowadays, and the percentage is reduced at present to something like 3 percent or 4 percent. What happened to that great big segment? . . . There is a large group that used to come in bleeding; most of them had a respiratory infection and had been sick for three or four days and then they would come in with massive hematemesis and melena. The workup would be negative, even with an upper gastrointestinal series . . . sometimes with a laparotomy. When we go back into that group we find that most of them had received aspirin, at least one dose, during the time that they had the upper respiratory infection—and this is the culprit. We have found in recent years that aspirin can cause bleeding. I caution you to shy away from operating on a child who has had aspirin, because of upper gastrointestinal bleeding. It does not have to be a high aspirin level; it does not have to be a toxic level; it does not have to be a handful of aspirin; the child does not have to have eaten it, it could be given rectally—and it could still be responsible for the gastrointestinal bleeding. It is an idiosyncrasy that is accentuated by the respiratory infection in some way that we do not completely understand, but it is a cause of a high incidence of gastrointestinal bleeding, particularly in the pediatric age group.

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